

Progressive Multiple Cranial Neuropathies as a Manifestation of ICANS Following CAR-T

Zachary Lazzari, Avi Singh Gandh, Samir Belagaje, Spencer Hutto

Objective

To describe a case of delayed onset multiple cranial neuropathies as manifestation of immune effector cell-associated neurotoxicity syndrome (ICANS) after chimeric antigen receptor T-cell (CAR-T) therapy for multiple myeloma.

Background

While ICANS following CAR-T is a well-reported complication, it classically presents with encephalopathy, headache, aphasia, tremors, seizures, and cerebral edema. Isolated unilateral facial neuropathies secondary to ICANS have been described, but multiple cranial neuropathies have not.

Design/Methods

N/A.

Results

A 75-year-old male presented with left facial nerve palsy 19 days after initiating CAR-T therapy for multiple myeloma. He developed fever, chills, and rigors 8 days post-CAR-T, which resolved with Tylenol (tocilizumab not administered). Contrasted brain MRI did not show abnormalities and his facial palsy was treated with prednisone 60 mg and Valtrex 1000 mg every 8 hours for 7 days. Left facial palsy persisted despite treatment and progressed to involve bilateral facial nerves and left cranial nerve VI by 31 days post-CAR-T. Specifically, his exam showed impaired abduction of left eye and twitches of orbicularis oculi without other facial movement (House-Brackmann grade VI bilaterally). Another contrasted MRI brain showed mild enhancement of bilateral facial nerves. Extensive serum and CSF testing was unremarkable. Initial treatment with prednisone 60 mg PO was ineffective. This was escalated to methylprednisolone 1 gram IV daily for 1 day, followed by 500 mg IV for 2 days, then a prolonged oral prednisone taper (39 days of therapy total). Concern for the impact of high dose steroids on CAR-T efficacy influenced treatment dose and duration. Anakinra was considered but not given. He had resolution of CN VI palsy at 2.5 months from onset (2 weeks after completed therapy), and moderate improvement of bilateral facial palsy 5.5 months from onset (3.5 months after completed therapy).

Conclusions

ICANS can present with progressive multiple cranial neuropathies and may benefit from treatment with corticosteroids.

Study Supported By: N/A.

Disclosure: Dr. Lazzari has nothing to disclose. Dr. Gandh has nothing to disclose. Dr. Belagaje has nothing to disclose. Dr. Hutto has nothing to disclose.

Post-Traumatic NMOSD Myelopathy in Octogenarians

Allyson Hamacher, Onilia Zorio, Alexandria Adamski, Marie Grill

Objective

N/A.

Background

Neuromyelitis Optica Spectrum Disorder (NMOSD), an inflammatory condition of the central nervous system with a predilection for optic nerve and spinal cord involvement, predominantly affects women in their 30s to 40s, though later onset disease may occur. Very late onset cases are rare and are more likely to present insidiously with extensive cord lesions. We present the cases of two octogenarian women with progressive lower extremity weakness days-weeks following fall/trauma who were ultimately diagnosed with NMOSD.

Design/Methods

N/A.

Results

The first patient presented at 88-years-old with progressive left leg weakness following a fall one month prior. Spine MRI demonstrated T2 hyperintense cord changes at T9-T11 with irregular enhancement and DWI changes. She presented again 12 months later with new progressive right leg weakness. Repeat MRI demonstrating new cord signal changes extending from T3-T6. Serum NMO/AQP4 antibodies were 1:10000; CSF titer was 1:16. She declined recommended disease modifying therapy (DMT) and had 2 subsequent attacks of longitudinally extensive transverse myelitis at 30 and 42 months. The second patient presented at 81-years-old with progressive left > right lower extremity weakness 3-days after ground-level fall. Spine MRI demonstrated T2 cord signal abnormality extending from C4-T12 with mild cord expansion and heterogeneous enhancement; as well as moderate to severe spinal stenosis at C4-C5 and C5-C6. Serum NMO/AQP4 antibodies were 1:100000; CSF titer was 1:512. Initiation of DMT was recommended.

Conclusions

Very-late-onset cases of NMOSD in patients over 80 are rare, whereas compressive/traumatic myelopathy is exceedingly more frequent in this age group. We present 2 cases of NMOSD in octogenarians who had experienced antecedent falls/trauma. Rare cases of multiple sclerosis post spinal cord injury have been reported with blood-brain barrier disruption suggested as putative mechanism. This may be of potential significance in patients with very late-onset NMOSD given increased incidence of traumatic/compressive myelopathy in this age demographic.

Study Supported By: N/A.

Disclosure: Ms. Hamacher has nothing to disclose. Onilia Zorio has nothing to disclose. Alexandria Adamski has nothing to disclose. Dr. Grill has nothing to disclose.

Driving Under the Cognitive Influence of COVID-19: Exploring the Impact of Acute SARS-CoV-2 Infection on Road Safety

Baran Erdik, Derek Homrich

Objective

This study evaluated the association between acute COVID-19 cases and the number of car crashes with varying COVID-19 vaccination rates, Long COVID rates, and COVID-19 mitigation strategies.

Background

The ongoing SARS-CoV-2 pandemic has led to significant concern over long-term post-infection sequelae, especially in the Neurologic domain. Long COVID symptoms, including cognitive impairments, could potentially impact activities requiring high cognitive function, such as driving. Despite various potential impacts on driving skills and the general prevalence of Long COVID, the specific effects on driving capabilities remain understudied.

Design/Methods

This study utilized a Poisson regression model to analyze data from 2020-2022, comparing aggregate car crash records and COVID-19 statistics. This model adjusted for population and included binary variables for specific months to account for stay-at-home orders. The correlation between acute COVID-19 cases and car crashes was investigated across seven states, considering vaccination rates and COVID-19 mitigation measures as potential confounders.

Results

Findings indicate an association between acute COVID-19 rates and increased car crashes with an OR of 1.5 (1.23-1.26 95%CI). The analysis did not find a protective effect of vaccination against increased crash risks, contrary to previous assumptions. The OR of car crashes associated with COVID-19 was comparable to driving under the influence of alcohol at legal limits or driving with a seizure disorder.

Conclusions

The study suggests that acute COVID-19, regardless of Long COVID status, is linked to an increased risk of car crashes presumably due to neurologic changes caused by SARS-CoV-2. These findings underscore the need for further research into the neuropsychological impacts of COVID-19. Further studies are recommended to explore the causality and mechanisms behind these findings and to evaluate the implications for public safety in other critical operational tasks. Finally, neurologists dealing with post-COVID patients, should remember that they may have an obligation to report medically impaired drivers.

Study Supported By: N/A.

Disclosure: Baran Erdik has nothing to disclose. Mr. Homrich has nothing to disclose.

Seeing Through the Blurry Vision of MOGAD vs NMOSD

Deeksha Barman, Swayam Prakash

Objective

This study aims to provide a comparative analysis of MOG-associated disorders (MOGAD) and Neuromyelitis Optica Spectrum Disorders (NMOSD), highlighting their distinct characteristics, diagnostic challenges, and implications for disease management.

Background

Optic neuritis (ON) is a common initial clinical presentation for both MOGAD and NMOSD, formerly categorized as seronegative variants of Multiple Sclerosis. Advances in diagnostic assays have identified specific antibodies, such as anti-MOG-IgG1 and anti-AQP4-IgG, enabling the reclassification of these disorders as separate entities.

Design/Methods

Case report.

Results

This report presents case studies of two female patients, aged 54 and 50, both exhibiting acute painful loss of vision (LOV). Clinical examinations, including visual acuity assessment, relative afferent pupillary defect (RAPD) evaluation and diagnostics like visual evoked potential (VEP) testing, and magnetic resonance imaging (MRI), showing optic nerve enhancements, supported the diagnoses of optic neuritis in both. Serological testing confirmed the presence of anti-MOG-IgG1 antibodies in one case and anti-AQP4-IgG antibodies in the other, distinguishing MOGAD from NMOSD. The patient with MOGAD, experiencing her first ON episode, showed significant improvement following steroid treatment. However, the NMOSD patient, with a history of recurrent ON episodes, did not respond adequately to steroids and required Rituximab therapy. Both patients remained relapse-free during follow-up.

Conclusions

This comparative analysis underscores the importance of distinguishing between MOGAD and NMOSD, elucidating their different prognostic trajectories and therapeutic outcomes. Unlike MS or MOGAD, NMOSD typically causes profound necrosis and cavitation affecting both gray and white matter rather than demyelination. Consequently, individuals with MOGAD commonly manifest favorable responses to treatment, however NMOSD patients often have recurrent episodes

necessitating intensified immunomodulatory regimens. Hence, timely serological testing guided by a high clinical suspicion facilitates accurate diagnosis and initiation of appropriate treatment, thereby improving disease outcomes.

Study Supported By: N/A.

Disclosure: Deeksha Barman has nothing to disclose. Dr. Prakash has nothing to disclose.

First Episode Psychosis and Status Epilepticus as Presenting Symptom of FLAIR-hyperintense Lesions in Anti-MOG-associated Encephalitis with Seizures (FLAMES)

Anza Zahid, Chao-Hsien Chen, Angela Salemi, Will Tanigaki, Sahithi Avva, Ashwin Achuthaprasad, Kathryn Boylan, Tanu Garg, Robert Smith

Objective

To report a novel entity with MOG-associated disease.

Background

FLAIR-hyperintense lesions in anti-MOG-associated Encephalitis with Seizures (FLAMES) phenotype is a novel entity described in young males with headaches, fluctuating weakness, and seizures. Herein, we describe a young patient with first onset psychosis and status epilepticus with MOG-associated FLAMES syndrome.

Design/Methods

Chart and literature review.

Results

A 23-year-old left-handed healthy man presented to an outside hospital with newly-onset headache followed by bizarre behavior, delusions, and visual hallucinations for 1 week. He had multiple seizure episodes during the hospitalization, necessitating treatment with three anti-seizure medications (ASM). Brain MRI revealed cortical and subcortical FLAIR hyperintensities in the left frontal gyrus suspicious for demyelinating etiology. Cerebrospinal fluid analysis showed pleocytosis and no oligoclonal banding. The autoimmune-encephalitis antibody and meningitis/encephalitis panel were negative. Despite empirical treatment with steroids and intravenous immunoglobulins, he continued to deteriorate clinically and was intubated. He was transferred to our institution for further care with modified Rankin score (mRS) of 4. Plasmapheresis was initiated for six sessions. The psychiatric symptoms and cognition improved significantly. No further seizures were recorded. Repeat Brain MRI demonstrated complete resolution of the previous lesions. His MOG titer was positive. Following plasmapheresis he received the induction therapy of rituximab. He was discharged from inpatient rehabilitation with mRS of 1 on maintenance rituximab therapy and ASM taper. At 4 month follow-up he denied any relapse.

Conclusions

MOG-associated disease can be misdiagnosed as viral encephalitis, autoimmune epilepsy, or substance-use disorder in young individuals presenting with atypical symptoms. FLAMES is a novel entity that has been described in the setting of positive MOG titers. Early recognition and treatment with plasmapheresis and anti-B cell immunomodulators is warranted. There is limited longitudinal data available on relapse associated with FLAMES. However, use of immunomodulating therapies such as rituximab and mycophenolate have shown benefit for sustained remission.

Study Supported By: N/A.

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